



Review article

Diesel exhaust exposure, its multi-system effects, and the effect of new technology diesel exhaust

Haley Reis^{b,1}, Cesar Reis^{a,b,*,1}, Akbar Sharip^c, Wenes Reis^a, Yong Zhao^{d,e,f}, Ryan Sinclair^g, Lawrence Beeson^{h,**}

^a Department of Preventive Medicine, Loma Linda University Medical Center, 24785 Stewart Street, Suite 204, Loma Linda, CA 92354, USA

^b Loma Linda University School of Medicine, 11175 Campus Street, Loma Linda, CA 92350, USA

^c Department of Occupational Medicine, Loma Linda University Medical Center, 328 East Commercial Road, Suite 101, San Bernardino, CA 92408, USA

^d School of Public Health and Management, Chongqing Medical University, Chongqing, China

^e Research Center for Medicine and Social Development, Chongqing Medical University, Chongqing, China

^f The Innovation Center for Social Risk Governance in Health, Chongqing Medical University, Chongqing, China

^g Center for Community Resilience, School of Public Health, Loma Linda University, Loma Linda, CA 92350, USA

^h Center for Nutrition, Healthy Lifestyle, and Disease Prevention, School of Public Health, Loma Linda University, Loma Linda, CA 92350, USA



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ABSTRACT

Exposure to diesel exhaust (DE) from vehicles and industry is hazardous and affects proper function of organ systems. DE can interfere with normal physiology after acute and chronic exposure to particulate matter (PM). Exposure leads to potential systemic disease processes in the central nervous, visual, hematopoietic, respiratory, cardiovascular, and renal systems. In this review, we give an overview of the epidemiological evidence supporting the harmful effects of diesel exhaust, and the numerous animal studies conducted to investigate the specific pathophysiological mechanisms behind DE exposure. Additionally, this review includes a summary of studies that used biomarkers as an indication of biological plausibility, and also studies evaluating new technology diesel exhaust (NTDE) and its systemic effects. Lastly, this review includes new approaches to improving DE emissions, and emphasizes the importance of ongoing study in this field of environmental health.

1. Introduction

Diesel exhaust (DE) is a complex mixture of hydrocarbons, gases, sulfur, and particulates produced during the combustion of diesel fuel (Hesterberg et al., 2011). It is considered an important source of ambient particulate matter (PM) in traffic-related air pollution. It has been estimated that 20–70% of PM is attributed to combustion-derived particles from traffic (Geller et al., 2005; Lanki et al., 2006), and up to 90% of PM in urban areas is traffic-related (Mazzarella et al., 2007). According to the study published by the Global Burden of Disease 2016 Risk Factors Collaborators, air pollution is among the leading top 10 risk factors for mortality in men and women in 2016 and has been considered a risk factor since 1990. In addition, occupational exposure from diesel exhaust engines was among the risks with an increase in its summary exposure value, a measure of exposure for each risk

(Collaborators, 2017). Moreover, according to the World Health Organization, > 7 million premature deaths annually are linked to air pollution, making it the largest single environmental health risk globally (WHO, 2014). In 2013, the International Agency for Research on Cancer (IARC) designated air pollution as a Group 1 carcinogen to humans based on the accumulating evidence regarding the relationship between particulate matter exposure and lung cancer risk (IARC, 2013). Other organ systems affected by the carcinogenic effects of DE include the central nervous system (CNS) and hematopoietic system (Danysh et al., 2015; Filippini et al., 2015). Non-carcinogenic changes are also associated with DE exposure, including retinal edema, conjunctivitis, bronchospasm, cough, systolic dysfunction, and changes in heart rate variability. Understanding the carcinogenic and systemic risks establishes the importance of reducing air pollution and improving the use of biomarkers to determine the level of exposure and its impact on

* Correspondence to: C. Reis, Department of Preventive Medicine, Loma Linda University Medical Center, 24785 Stewart Street, Suite 204, Loma Linda, CA 92354, USA.

** Corresponding author.

E-mail addresses: cesarreis@hotmail.com (C. Reis), lbeeson@llu.edu (L. Beeson).

¹ Authors contributed equally to this work.

Table 1
Traditional diesel exhaust (DE) and new technology diesel exhaust.

	TDE	NTDE
Carbon	41% (primarily elemental carbon)	13% elemental carbon + 30% organic carbon
Elements with sulfur/sulfate	14% (sulfate and water)	57%
Unburnt fuel	25%	0%
Ash and other	13%	0%
Unburnt fuel	7%	0%
Elements without sulfur	0%	4%

This table allows comparisons of major components between pre-2007 Diesel exhaust and post-2007 Diesel exhaust (Hesterberg et al., 2011). After institution of the NTDE the amount of carbon present in the exhaust has decreased drastically.

cellular, oxidative, and inflammatory processes.

The Environmental Protection Agency (EPA) placed standards in 2007 to protect against DE exposure and emissions. US EPA 2007/2010 Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements generated a movement towards developing enhanced technology to emit 90% less concentrated particulate constituents compared to traditional DE. The new standards are estimated to reduce PM emissions by 110,000 tons each year and toxic air pollutants such as benzene by 17,000 tons in the U.S. annually (MECA, 2017). The newly designed diesel engines and utilization of new technology diesel exhaust (NTDE) decreases the carcinogenic risk and reduces the physiological impact of particulate matter (Costantini et al., 2016). Animal studies investigate the effects of NTDE, specifically its effects on the respiratory and cardiovascular system, and compare these outcomes to former studies that used traditional diesel exhaust (TDE). According to preliminary toxicological data, significant chemical distinctions exist between NTDE particulate and DE particulate matter from pre-2007 diesel technology, including differences in biological responses. The presence of organic carbon and a higher percentage of elements with sulfur set NTDE apart from traditional DE (Table 1). Khalek et al., as part of the Advanced Collaborate Emissions Study (ACES), measured PM emissions and three other regulated emissions (carbon monoxide, non-methane hydrocarbon, and nitric oxide) on U.S.

Environment Protection Agency (EPA)- compliant heavy-duty diesel engines. The emissions were well below the 2007 standards, with only 13% of PM being elemental carbon, or soot. An 86% total reduction in PM emissions was found compared to a 2004-technology engine (Khalek et al., 2011).

This review brings an important perspective on DE exposure. It takes on a multi-system approach in discussing DE and the consequences of both acute and chronic exposure. This review emphasizes the importance of identifying the specific components that make DE dangerous, studying NTDE and its epidemiological effects, and carefully evaluating potential alternative fuel options and whether they too pose a threat to biological processes.

2. Methods

We searched the PubMed database for literature on diesel exhaust and its multi-system effects, including epidemiology and pathogenic mechanisms. The systems included in this review were chosen based on the amount of literature discussing the organ system, and the overall burden of DE related to that organ system. Our systems were also based on the studies we found that measured biomarkers. Most of the biomarker studies focused on the respiratory and cardiovascular systems and are included in the respective sections. We also chose CNS and hematopoietic because DE is implicated in causing carcinogenic effects to these body systems. Finally, the visual and renal systems provided information on areas of human physiology that are not normally considered troubling upon exposure to DE, yet studies find pathogenesis in these system upon DE exposure. The inflammatory system, though not a section of its own, is incorporated into the pathogenesis of diseases related to DE exposure and is discussed throughout the review. Most of the organ system searches had a filter to include studies that were published within the last five years, except for the respiratory system (Fig. 1). This portion of the research included studies from within the last 10 years to fully elucidate the pathogenesis of DE on this organ system (Table 1).

Most of the studies included in this review were published after the EPA standards were updated in 2007 to decrease DE emissions, and the term DE was used to describe the type of exhaust being studied. The specific components of the DE were not described, making it difficult to

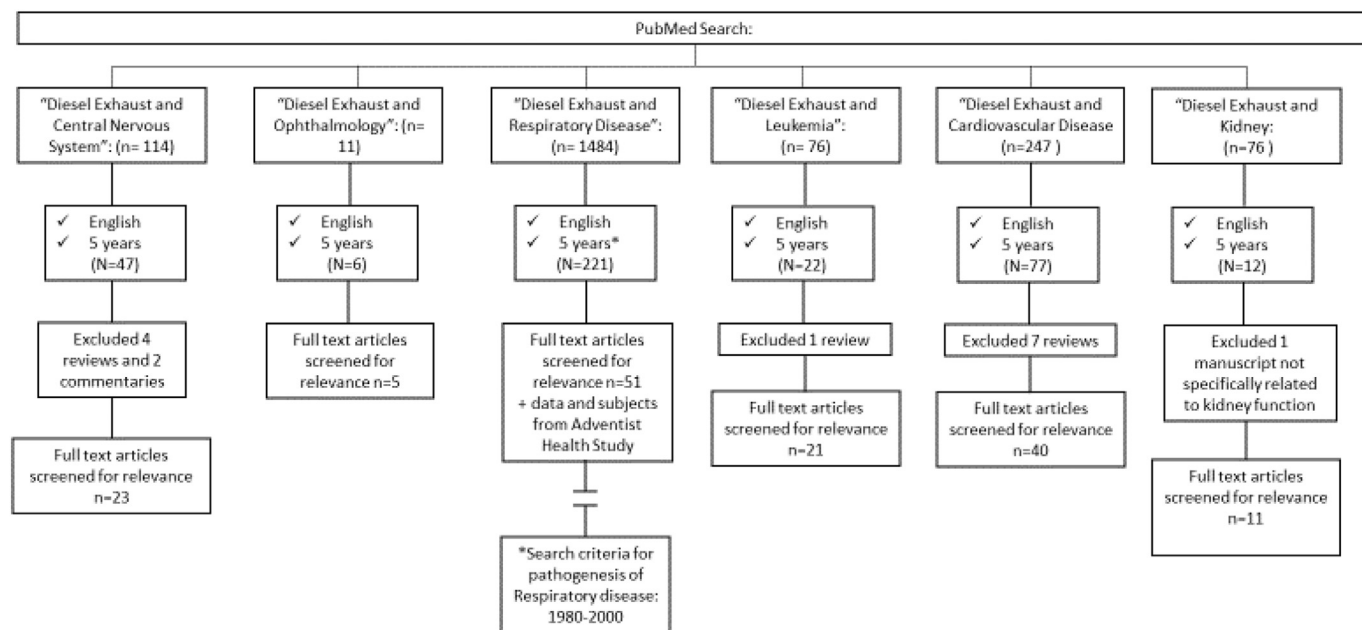


Fig. 1. Schematic of search strategy.

This figure includes our search strategy for diesel exhaust and its effects on multiple organ systems. After checking papers in English published over the last 5 years, we excluded reviews and commentaries and kept relevant papers.

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